Arachnoiditis is chronic inflammation of the arachnoid layer of the meninges, which consists of trabeculae, a mesh of interwoven collagen fibrils resembling tissue paper.

These secrete spinal fluid, which circulates through the cerebrospinal axis and is absorbed through the arachnoid villi in the brain.

The initial phase of the inflammatory process involves influx of white blood cells in response to an insult to the subarachnoid space, such as blood (trauma, surgery), foreign substance (dye, etc) or infectious agent (e.g. meningitis).

This is initiated via the action of cytokines. There is infiltration by macrophages and mesenchymal cells; the latter transform into fibroblasts, which lay down collagen.

Usually the fibrinolytic process, which breaks down excess scar tissue, limits this, but in arachnoiditis the scar tissue continues to form.

Authors such as Jayson ([i]) have suggested that there may be a defect in the fibrinolytic pathway.

In the first stage involves radiculitis and the adjacent blood vessel hyperaemia. The subarachnoid space disappears. Deposition of collagen fibrils begins.

In the second stage, (arachnoiditis) the scar tissue increases, and the nerves become adherent to each other and the dura.

The third stage, (adhesive arachnoiditis), involves complete encapsulation of the nerve roots. The subsequent compression causes them to atrophy.

The scarring prevents the arachnoid from producing spinal fluid in that area.

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[i] Jayson MI, Keegan A, Million R, Tomlinson I *Lancet* 1984 Nov 24; 2(8413): 1186-7 A fibrinolytic defect in chronic back pain syndromes.